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Intra- and Inter-tooth Analysis of Hypoplastic and Hypocalcified Enamel Defects

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INTRODUCTION

Dental enamel defects resulting from systemic metabolic stress, rather than from genetic or traumatic causes, are referred to as "developmental" defects and indicate non-specific physiological stress during childhood. Two categories of defects are commonly descri-bed: hypoplasia and hypocalcification (Fédération Dentaire Internationale, 1982; Suckling et al., 1989; Weinmann et al., 1945), with each linked to a disturbance during a specific phase of enamel development and having a unique process of defect creation. Although the frequency of hypoplastic defects has been used to determine the effect of culture change, such as the arrival of Europeans in the New World; the rise of industrialization and urbanization during the 18th and 19th centuries; and the collapse of ancient civilizations, on past health (e.g., Santos and Coimbra, 1999; Wood, 1996; Wright, 1997), the validity of enamel hypocalcification as an indicator of non-specific stress has been que-stioned. At least one study has shown that althou-gh enamel hypoplasia is related to socioeconomic or nutritional status, enamel hypocalcification is not (Goodman, 1991). The purpose of this study, therefore, is to investigate the possibility that these two defect classes may not represent the same stresses or life conditions, by comparing their frequency and distribution within a dental sample from the ancient Mesopotamian site of Tell Leilan: if both defect classes represent non-specific stress then a similar defect pattern should be evident.

Tell Leilan is located on the Khabur plain of northeastern Syria and is one of the largest sites in northern Mesopotamia. Historical and archaeological investigations have revealed a dynamic culture history during which settlement size and density, social organization, and climate were affected (Weiss, 1990; Weiss and Bradley, 2001; Weiss et al., 1993). As a consequence, some individuals likely were stressed by malnutrition and/or infectious disease, resulting in defective dental enamel.

MATERIALS AND METHODS

The Tell Leilan dental sample was obtained from the skeletal remains of 23 individuals, ranging in age from prenatal to approximately 15 years, that were excavated between 1979 and 1989 (see Weiss, 1985, 1990; Weiss and Bradley, 2001; Weiss et al., 1993) and which date to the mid-3rd millennium BC. Three factors determined the selection of dental specimens to be analyzed: 1) teeth with more than slight wear (stage 2; Smith, 1984) were excluded since moderate to severe attrition would destroy evidence of defects in the incisal/occlusal third of the crown; 2) teeth with other antemortem conditions such as caries or unusual wear patterns were excluded; 3) teeth revealing postmortem damage such as chipping, cracking or enamel loss within one of the three areas were excluded. This yielded a study sample of 153 permanent teeth. After calculus deposits were scored (following Buikstra and Ubelaker, 1994:56), teeth were cleaned manually and consolidant that had been applied in the field was removed with acetone. The type and location of hypoplastic and hypocalcified defects were observed with the unaided eye and verified with a hand lens and a light microscope if necessary. All permanent tooth types were examined so that all age ranges of dental development could be included. The left antimere of each tooth type was chosen for analysis but if the left antimere was not present or scorable then the right antimere was used.

Defects were recorded as to class of defect, type of defect, and position of defect by surface and by tooth-third location (Table 1) according to the Developmental Defects of Enamel (DDE) index (Buikstra and Ubelaker, 1994; Fédération Dentaire Internationale, 1982). In order to provide data comparable to other studies, the location of defects was recorded for the facial surface only. A defect is defined as any irregularity or abnormality in the dental enamel. Normal enamel is usually white to cream in color and translucent, and the crown surface is smooth. *Enamel hypoplasia* is

Table 1: Descriptors of dental enamel defects (based on Buikstra and Ubelaker 1994; Fédération Dentaire Internationale 1982)

Type of enamel	*Type of defect*	*Affected crown surface*	*Defect location on tooth crown*
tooth not present	pitting	lingual	cervical third
tooth unscorable	horizontal grooves (LEH)	facial	middle third
normal enamel	focal loss of enamel	both surfaces	incisal/occlusal third
hypoplastic enamel	diffuse opacity		cervical and middle thirds
hypocalcified enamel	demarcated opacity		middle and incisal thirds
			entire tooth crown

defined as any reduction in the quantity of dental enamel and is the result of a systemic disruption during the secretory phase of enamel development. Enamel hypoplasia can appear as a single pit or several pits, as a focal loss of enamel, or as horizontal grooves, also known as "linear enamel hypoplasia" or LEH. These may appear on the lingual or facial tooth surface or may circumscribe the tooth. The etiological and microstructural differences between the different types of hypoplastic lesions are not well understood (see Hillson and Bond 1997 for a discussion).

In contrast to hypoplasia, *hypocalcification* results in enamel of lower quality due to a systemic insult during the mineralization phase of enamel development. It alters the enamel from translucent to opaque or discolored, which may be exhibited as either demarcated/discrete or diffuse. Demarcated opacities are lesions separated from normal enamel by a definite boundary, while diffuse opacities are patchy or cloudy and lack well-defined margins.

RESULTS

As presented in Table 2 and Table 3 79% of

Table 2: Enamel defect frequencies by permanent tooth type

	Hypoplasia					*Hypocalcification*				
	N_o	N_a	%	*n*	n/N_a	N_o	N_a	%	*n*	n/N_a
Maxillary										
I1	9	6	67	6	1.00	9	7	78	13	1.86
I2	8	7	88	8	1.14	8	7	88	9	1.29
C	8	7	88	10	1.43	8	6	75	8	1.33
PM1	11	10	91	12	1.20	11	8	73	9	1.13
PM2	12	10	83	12	1.20	12	10	83	10	1.00
M1	10	8	80	8	1.00	10	8	80	8	1.00
M2	7	5	71	5	1.00	7	6	86	7	1.17
M3	6	6	100	8	1.33	6	5	83	8	1.60
Subtotal	71	59	83	69	1.17	71	57	80	72	1.26
Mandibular										
I1	6	3	50	3	1.00	6	4	67	4	1.00
I2	8	6	75	7	1.17	8	4	50	4	1.00
C	11	10	91	12	1.20	11	8	73	10	1.25
PM1	12	11	85	13	1.18	13	8	62	8	1.00
PM2	13	10	77	11	1.10	13	7	54	7	1.00
M1	11	7	64	7	1.00	11	6	55	6	1.00
M2	11	7	64	11	1.57	11	6	55	7	1.17
M3	9	8	89	11	1.38	9	5	56	6	1.20
subtotal	82	52	76	75	1.21	82	48	58	52	1.08
Total	153	121	79	144	1.19	153	105	68	124	1.18

N_o = number of scorable teeth
N_a = number of teeth with one or more hypoplastic or hypocalcified defects
% = frequency of hypoplastic or hypocalcified teeth (number of defective teeth divided by the total number of teeth scored)
n = total number of hypoplastic or hypocalcified defects
n/N_a = defect ratio (number of defects divided by the total number of defective teeth)

the teeth were hypoplastic, revealing a total of 144 defects; and 68% of the teeth exhibited enamel hypocalci-fication in the form of 124 defects. When a standardizing defect ratio is calculated by dividing the number of hypoplastic or hypo-calcified defects by the total number of defective teeth, the number of stress episodes per defective tooth is approximately equal: 1.19 for hypoplasia and 1.18 for hypocalcification. However, although the maxillary and mandibular dentitions are affected almost equally by hypoplasia, the maxillary teeth show more hypocalcification than do the mandibular teeth.

Although the defect ratio points to a similar overall stress profile for hypoplasia and hypocalcification, the inter-tooth frequencies and defect ratios differ for each tooth type (Table 2). If considered by the frequency of defective teeth, the most hypoplastic tooth type is the maxillary third molar (100%), followed by the mandibular canine (91%) and the maxillary first premolar (91%). The least defective tooth type for hypoplasia is the mandibular central incisor (50%). If considered by defect ratio, the mandibular second molar is the most hypoplastic at 1.57 defects per affected tooth. Five teeth have defect ratios of 1.00.

The most hypocalcified tooth types, as indicated by the frequency of defective teeth, are the maxillary second incisor (88%) and second molar (86%) (Table 2). If considered by defect ratio, the maxillary central incisor is the most hypocalcified, at 1.86 defects per affected tooth. The least hypocalcified tooth types are the mandibular lateral incisor (50%) and the second premolar (54%), and seven teeth have defect ratios of (1.00). Eighty-two per cent of the hypoplastic defects were linear in form (LEH) and 95% of the hypocalcified defects were diffuse.

Both classes of defects were most often present on the facial surface (Table 4). The majority of hypoplastic defects involved only the facial surface (65%), followed by defects incorporating both surfaces (28%) and a few isolated on the lingual surface (7%) (Table 5). In contrast, the majority of hypocalcified defects incorporated both the lingual and facial surfaces (45%), followed by defects restricted to the facial surface (40%).

Hypoplastic defects are most often found in the cervical or middle thirds or involve both the cervical and middle thirds (Table 6). Similarly, hypocalcified defects were most prevalent in the cervical third, or involved both the cervical and middle thirds (Table 7). While all classes of teeth exhibit hypocalcified defects at the cervix, the posterior teeth report the only defects on the middle third. Although rare, the maxillary anterior teeth did exhibit hypocalcification in the occlusal third, as well as hypocalcification that involved the entire crown surface.

DISCUSSION

Intra- and inter-tooth variability in defect frequency and distribution are believed to reflect relative sensitivity or susceptibility of different tooth types and tooth areas to physiological insult. In a review of past studies, Goodman and Armelagos (1985) test the assumption that different tooth types respond uniformly to stress and conclude that defect frequency varies substantially between tooth types as well as within individual tooth crowns. Three theories have been proposed to explain intra- and inter-tooth variation in hypoplastic and hypocalcified defect expression: 1) a hypoplastic defect is formed only when the magnitude of stress for a given tooth type reaches a critical threshold (Goodman and Rose, 1990); 2) variation in the rate of enamel deposition between and within tooth types contributes to variation in defect expression (Condon and Rose, 1992; Fearne et al,. 1994; Needleman et al., 1991; Suga, 1989); and 3) crown geometry, namely appositional versus imbrica-tional zones of crown growth and spacing bet-ween developmental layers, is related to hypoplastic expression (Guatelli-Steinberg and Lukacs, 1998; Hillson and Bond, 1997).

Intra-tooth variation in the Tell Leilan sample reveals a higher prevalence of hypoplastic and hypocalcified defects on the facial surface and in the cervical and middle thirds of each tooth type, in agreement with previous studies (e.g. Condon and Rose, 1992; Goodman and Armelagos, 1985; Needleman et al., 1991; Smith and Peretz, 1986). In contrast, inter-tooth variation is inconsistent. Most studies have found the anterior teeth, especially the maxillary anterior teeth, to be more hypoplastic and hypocalcified than are the posterior teeth (Al-Abbasi and Sarie´, 1997;

Table 3: A comparison of the frequency and intertooth distribution of hypoplastic and hypocalcified defect types, including both lingual and labial surface defects.

	Hypoplasia							*Hypocalcification*				
		Pitting		*LEH*		*Local loss*			*Diffuse*		*Demarcated*	
	N_h	*n*	*%*	*n*	*%*	*n*	*%*	N_o	*n*	*%*	*n*	*%*
Maxillary												
I1	6	0	-	6	100	0	-	13	13	100	0	-
I2	8	0	-	5	6	3	37	9	9	100	0	-
C	10	0	-	9	90	1	10	8	8	100	0	-
PMI	12	1	8	11	92	0	-	9	9	100	0	-
PM2	12	1	8	10	84	1	8	10	10	100	0	-
M1	8	1	12	6	75	1	12	8	8	100	0	-
M2	5	0	-	4	80	1	20	7	6	86	1	14
M3	8	2	25	6	75	0	-	8	6	75	2	25
Mandibular												
I1	3	0	-	3	100	0	-	4	4	100	0	-
I2	7	1	14	5	71	1	14	4	4	100	0	-
C	12	0	-	10	83	2	17	10	9	90	1	10
PM1	13	2	15	11	85	0	-	8	8	100	0	-
PM2	11	1	9	10	91	0	-	7	7	100	0	-
M1	7	0	-	7	100	0	-	6	6	100	0	-
M2	11	3	27	8	73	0	-	7	6	86	1	14
M3	11	4	36	7	64	0	-	6	5	83	1	17
Total	144	16	11	118	82	10	7	124	118	95	6	5

N_h = total number of hypoplastic defects per tooth type
N_o = total number of hypocalcified (opaque) defects per tooth type
n = number of hypoplastic or hypocalcified defects per tooth third or tooth third combination
% = frequency of hypoplastic or hypocalcified defects per tooth third or tooth third combination

Table 4: A comparison of hypoplastic and hypocalcified defect frequency and inter-tooth distribution by affected surface. Defects incorporating both the lingual and facial surfaces are counted as one lingual plus one facial defect.

	Hypoplasia					*Hypocalcification*				
		Lingual		*Facial*			*Lingual*		*Facial*	
	N_h	*n*	*%*	*n*	*%*	N_o	*n*	*%*	*n*	*%*
Maxillary										
I1	6	0	-	6	100	15	6	40	9	60
I2	9	1	11	8	89	15	6	40	9	60
C	11	3	11	8	73	13	6	46	7	54
PMI	15	4	27	11	73	11	6	55	5	45
PM2	17	6	35	11	65	16	8	50	8	50
M1	10	2	20	8	80	12	4	33	8	67
M2	7	2	29	5	71	9	4	44	5	56
M3	9	3	33	6	67	9	4	44	5	56
Mandibular										
I1	3	0	-	3	100	6	2	33	4	67
I2	7	1	14	6	86	5	1	20	4	80
C	17	5	29	12	71	16	6	37	10	63
PM1	19	7	37	12	63	14	6	43	8	57
PM2	18	8	44	10	56	13	7	54	6	46
M1	8	1	12	7	88	9	3	33	6	67
M2	13	4	31	9	69	10	4	40	6	60
M3	16	5	31	11	69	7	2	29	5	71
Total	185	52	28	133	72	180	75	42	105	58

N_h = total number of hypoplastic defects per tooth type
N_o = total number of hypocalcified (opaque) defects per tooth type
n = number of hypoplastic or hypocalcified defects present per surface
% = frequency of hypoplastic or hypocalcified defects present per surface

Table 5: A comparison of the frequency and inter-tooth distribution of hypoplastic and hypocalcified defects involving the lingual, facial, or both surfaces.

	Hypoplasia							*Hypocalcification*						
		Lingual		*Facial*		*Both*			*Lingual*		*Facial*		*Both*	
	N_h	*n*	*%*	*n*	*%*	*n*	*%*	N_o	*n*	*%*	*n*	*%*	*n*	*%*
Maxillary														
I1	6	0	-	6	100	0	-	13	4	31	7	54	2	15
I2	8	0	-	7	87	1	13	9	0	-	3	33	6	67
C	10	2	20	7	70	1	10	8	1	13	2	25	5	62
PMI	12	1	8	8	67	3	25	9	4	45	3	33	2	22
PM2	12	1	8	6	50	5	42	10	2	20	2	20	6	60
M1	8	0	-	6	75	2	25	8	0	-	4	50	4	50
M2	5	0	-	3	60	2	40	7	2	29	3	42	2	29
M3	8	2	25	5	62	1	13	8	3	37	4	50	1	13
Mandibular														
I1	3	0	-	3	100	0	-	4	0	-	2	50	2	50
I2	7	1	14	6	86	0	-	4	0	-	3	75	1	25
C	12	0	-	7	58	5	42	10	0	-	4	40	6	60
PM1	13	1	8	6	46	6	46	8	0	-	2	25	6	75
PM2	11	1	10	3	30	7	70	7	1	14	0	-	6	86
M1	7	0	-	6	86	1	14	6	0	-	3	50	3	50
M2	11	2	18	7	64	2	18	7	1	14	3	43	3	43
M3	11	0	-	6	54	5	46	6	1	17	4	66	1	17
Total	144	10	7	93	65	41	28	124	19	15	49	40	56	45

N_h = total number of hypoplastic defects per tooth type
N_o = total number of hypocalcified (opaque) defects per tooth type
n = number of hypoplastic or hypocalcified defects involving the surface(s)
% = frequency of hypoplastic or hypocalcified defects involving the surface(s)

Table 6: Frequency and inter-tooth distribution by tooth third of hypoplastic defects present on the labial surface.

		Cervical		*Middle*		*Incisal*		*Cervical & middle*		*Middle & incisal*		*Entire tooth crown*	
	N	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*
Maxillary													
I1	6	0	-	2	33	0	-	4	67	0	-	0	-
I2	8	1	12	4	50	1	12	2	25	0	-	0	-
C	8	2	25	1	12	0	-	5	63	0	-	0	-
PMI	11	3	27	7	64	1	9	0	-	0	-	0	-
PM2	11	4	36	4	36	1	10	2	18	0	-	0	-
M1	8	4	50	4	50	0	-	0	-	0	-	0	-
M2	5	3	60	1	20	0	-	1	20	0	-	0	-
M3	6	5	83	0	-	0	-	1	17	0	-	0	
Mandibular													
I1	3	1	33	2	67	0	-	0	-	0	-	0	-
I2	7	1	14	4	57	0	-	2	29	0	-	0	-
C	12	2	17	4	33	0	-	5	42	0	-	1	8
PM1	12	6	50	5	42	0	-	0	-	0	-	1	8
PM2	10	3	30	5	50	0	-	2	20	0	-	0	-
M1	8	5	63	1	12	0	-	1	12	0	-	1	12
M2	9	3	33	5	56	0	-	1	11	0	-	0	-
M3	11	4	36	2	17	4	36	1	9	0	-	0	-
Total	135	47	35	51	38	7	5	27	20	0	-	3	2

N = total number of hypoplastic defects per tooth type
n = number of hypoplastic defects per tooth third or combination of tooth thirds
% = frequency of hypoplastic defects per tooth third or combination of tooth thirds

Table 7: Frequency and intertooth distribution by tooth third of hypocalcified defects present on the facial surface.

		Cervical		*Middle*		*Incisal*		*Cervical & Middle*		*Middle & Incisal*		*Entire Tooth Crown*	
	N	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*
Maxillary													
I1	9	2	22	0	-	2	22	2	22	1	12	2	22
I2	9	4	44	0	-	2	22	2	22	0	-	1	12
C	7	2	29	0	-	1	13	2	29	0	-	2	29
PMI	5	1	20	1	20	0	-	2	40	0	-	1	20
PM2	8	3	38	1	12	0	-	4	50	0	-	0	-
M1	8	6	75	1	12	0	-	1	12	0	-	0	-
M2	5	4	80	0	-	0	-	1	20	0	-	0	-
M3	5	3	60	1	20	0	-	1	20	0	-	0	-
Mandibular													
I1	4	3	75	1	25	0	-	0	-	0	-	0	-
I2	4	3	75	1	25	0	-	0	-	0	-	0	-
C	10	2	20	1	10	1	10	5	50	0	-	1	10
PM1	8	4	50	1	12	0	-	2	25	1	12	0	-
PM2	6	4	67	2	33	0	-	0	-	0	-	0	-
M1	6	5	83	1	17	0	-	0	-	0	-	0	-
M2	6	5	83	0	-	0	-	1	17	0	-	0	-
M3	5	2	40	2	40	0	-	1	20	0	-	0	-
Total	105	53	50	13	12	6	6	24	23	2	2	7	7

N = total number of hypocalcified defects per tooth type
n = number of hypocalcified defects per tooth third or tooth third combination
% = frequency of hypocalcified defects per tooth third or tooth third combination

Cucina et al., 1996; Goodman and Armelagos, 1985; Needleman et al., 1991; Smith and Peretz, 1986; Wright, 1997). By contrast, in the Tell Leilan sample neither the maxillary nor mandibular dentitions exhibit differences in frequency of hypoplasia between the anterior and posterior teeth. More specifically, although the maxillary central incisor and mandibular canine are usually found to be the most hypoplastic tooth types (e.g. Al-Abbasi and Sarie,´ 1997; Cucina et al., 1996; Goodman and Armelagos, 1985), in the Tell Leilan sample the maxillary central incisor is the least defective. The maxillary third molar is the most hypoplastic in the Leilan sample, although the significance of this is difficult to assess since most studies do not include third molars. Those that do, however, have not found high frequencies of hypoplasia in the posterior teeth (Al-Abbasi and Sarie´, 1997; Cucina et al., 1996). If inter-tooth susceptibility is assessed by examining the defect ratio then the maxillary canine and the mandibular second molar exhibit the highest number of stress episodes. Again, this contradicts previous findings. Following the same calculation, Goodman and Armelagos (1985) found that the maxillary central incisor was the tooth most affected by enamel defects.

In contrast to hypoplastic defects, enamel opacities are most frequently found on the facial surface of the maxillary teeth, with the lateral incisor being the most frequently hypocalcified tooth type. This frequency and distribution is consistent with epidemiological data on hypocalcified defect expression (Forrest and James, 1965; Murray and Shaw, 1979; Nevitt et al., 1963). If an inter-tooth comparison is based on the defect ratio, then more defects are found in the maxillary anterior teeth as compared to the posterior, consistent with other studies (e.g., Needleman et al., 1991), and the maxillary central incisor exhibits the most hypocalcified defects.

In contrast to the conclusions from the analysis of hypoplasia, hypocalcification data suggest a different pattern of stress; namely, different inter-tooth frequencies of defective teeth and different numbers of stress episodes. Several factors may explain these conflicting results. First, in a discussion of the limited number of anthropological studies analyzing hypocalcification as an indicator of non-specific stress,

Hillson (1996) notes that the concern of potential post-mortem/taphonomic influences, which may affect enamel color or translucency, may deter their use. Enamel hypoplasia has long been considered significantly more resistant to alteration in the burial environment; Rose et al. (1985:285) note that many studies have found enamel unaltered in the burial environment and, subsequently, hypoplastic defects have been considered the result of metabolic disturbances for some time. However, post-mortem conditions in the burial environment may involve leaching of the mineral content of teeth. Since enamel hypocalcification results in less mineralized enamel with a higher organic content (Crenshaw and Bawden, 1984; Suga, 1989), distinguishing between post-mortem and ante-mortem causes of lower mineralization may be of concern. Furthermore, as Needleman and co-workers (1991:213) note, opacities may be easier to detect or even artificial on dried enamel. Suckling et al. (1989:226) also found that diffuse opacities are widely distributed over the tooth crown when the enamel is dry.

Second, antemortem conditions can also affect the frequency and distribution of enamel hypocalcification. Needleman and co-workers (1991:213) note the potential of dental calculus to decalcify enamel and produce "white spot" decalcification, which can imitate hypocalcification. As Croll (1991:22, 26) explains, enamel hypocalcification is *developmental* since it is the result of a disturbance during the mineralization phase of enamel development. Enamel decalcification, on the other hand, is *acquired* and occurs when the organic acids produced by dental plaque etch the mineral content from the enamel surface. Acquired enamel decalcification is usually seen in the cervical region of all teeth, which is a common site for dental calculus, and for this reason, Needleman and co-workers excluded opaque defects found in this region from their analyses. Initial preparation of the Tell Leilan dental sample included scoring and removal of dental calculus. If dental calculus did influence hypocalcification frequency and distribution, this would explain the higher frequency of defects present on the lingual surface, as well as defects involving both the lingual and facial surfaces.

CONCLUSION

A comparative analysis of hypoplastic and hypocalcified defect frequency and distribution in the Tell Leilan dental sample suggest different patterns of non-specific stress. Although intra-tooth variation is consistent with previous studies, inter-tooth defect patterns differ between hypoplasia and hypocalcification. Two factors may be influencing this differential patterning: postmortem alteration can create pseudo-opacities; and antemortem conditions, such as dental calculus, can produce opacities that are *acquired* rather than *developmental*. Due to contrasting evidence from the analysis of enamel hypocalcification as compared to hypoplasia, as well as in light of possible non-developmental factors acting on prevalence and distribution, the use of enamel hypocalcification as an indicator of non-specific stress should be reconsidered.

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KEY WORDS Hypoplasia. Hypocalcification. Mesopotamia.

ABSTRACT A comparison of the frequency and distribution of hypoplastic and hypocalcification defects in 153 permanent teeth reveals two different patterns. Although intra-tooth variation is consistent with the results of previous studies, inter-tooth patterns of hypoplasia and hypocalcification differ, and suggest that it may be difficult to distinguish developmental from acquired opacities. Two factors may be responsible for acquired opacities: postmortem alteration and antemortem conditions, such as dental calculus or localized trauma. These results indicate that the use of enamel hypocalcification as an indicator of non-specific stress, and hence as a proxy for health of past populations, should be reconsidered.

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